10/532,775

Boyle

AN 94123361 MEDLINE
DN PubMed ID: 8293487
TI Secondary osteoporosis.
AU Boyle I T
CS University Department of Medicine, Glasgow Royal Infirmary, UK.
SO Bailliere's clinical rheumatology, (1993 Oct) Vol. 7, No. 3, pp. 515-34.
Ref: 85
Journal code: 8805770. ISSN: 0950-3579.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 199403
ED Entered STN: 14 Mar 1994
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AB Osteoporosis with attendant increased fracture risk is a common complication of many other diseases. Indeed, almost all chronic diseases make some impact on life-style, usually by restricting physical activity and hence reducing the anabolic effect of exercise and gravitational strains on the skeleton. Restricted appetite and modified gastrointestinal tract function is another commonplace finding that has an impact on bone nutrition and synthesis, as on other systems. Sex hormone status is of particular importance for the maintenance of the normal skeleton, and the postmenopausal woman is at particular risk for most causes of secondary osteoporosis. In dealing with secondary osteoporosis in the hypo-oestrogenic woman, the question of giving hormone replacement therapy in addition to other disease-specific therapy should always be considered, as, for example, in a young amenorrhoeic woman with Crohn's disease. Similarly, in hypogonadal men the administration of testosterone is useful for bone conservation. The wider availability of bone densitometry ought to make us more aware of the presence of osteoporosis in the many disease states discussed above. This is particularly important as the life span of such patients is now increased by improved management of the underlying disease process in many instances. Even in steroid-induced osteoporosis--one of the commonest and most severe forms of osteoporosis--we now have some effective therapy in the form of the bisphosphonates and other anti-bone-resorbing drug classes. The possibility of prophylaxis against secondary osteoporosis has therefore become a possibility, although the very long-term effects of such drug regimens are still unknown. In some situations, such as thyrotoxicosis, Cushing's syndrome and immobilization, spontaneous resolution of at least part of the osteoporosis is possible after cure of the underlying problem. The shorter the existence of the basic problem, the more successful the restoration of the skeleton appears to be. A useful credo for clinicians with respect to secondary osteoporosis is: to think of it; to use specific therapy for the underlying disease; to reduce or remove completely any relevant drug or toxic material; to optimize physical activity and general nutrition; to treat hypogonadism if present and feasible; and to consider the use of specific anti-bone-resorbing or other bone active drugs.

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Frost

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DN PubMed ID: 9567365
TI Osteoporosis treatment: quo vadis? (A brief overview).
AU Frost H M
CS Department of Orthopaedic Surgery, Southern Colorado Clinic, Pueblo 91991, USA.
SO Medicina, (1997) Vol. 57 Suppl 1, pp. 119-26. Ref: 17
Journal code: 0204271. ISSN: 0025-7680.
CY Argentina
DT Journal; Article; (JOURNAL ARTICLE).
General Review; (REVIEW)
LA English
FS Priority Journals
EM 199806
ED Entered STN: 18 Jun 1998
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AB What we formerly called osteoporosis includes four conditions with an osteopenia: A) osteopenias usually due to mechanical disuse, where injuries cause fractures, and in limb bones more than the spine; B) osteopenias with such fragile bone that normal activities instead of injuries can cause fractures and/or bone pain, and in the spine more than limb bones; C) a group that combines features of (A) and (B); D) temporary osteopenias while major fractures, burns or other injures heal. If belatedly, we now realize our past failure to view those conditions as separate entities compromised many past studies of the prevalence, diagnosis and ways to prevent and cure each of them. That failure also compromised many past explanations of the nature, pathogenesis and natural course of "osteoporosis", and much of the related research. This caused some confusion as well as controversies about illusory instead of genuine issues. Controlling existing osteoblasts and osteoclasts with drugs has not prevented or cured those conditions. That will require controlling the modeling drifts and remodeling BMUs that create those cells. Modeling can increase bone mass and strength, remodeling can conserve or reduce them, and neither can provide the other's functions. During normal mechanical usage modeling is OFF and remodeling works in its "conservation mode" to keep existing bone. In disuse, modeling stays OFF while remodeling works in its "disuse mode" to remove bone and cause an osteopenia. Most natural nonmechanical agents (Table 1) can help or hinder those mechanical responses, but cannot duplicate or override them. Wrist and hip fractures from falls cause the most serious problems associated with these conditions. Those fractures begin in the cortex of epiphyseal-metaphyseal regions of limb bones. They never begin in trabecular bone and rarely in the shafts of long bones. They never begin in the trabecular bone and rarely in the shafts of long bones. Since a bone's strength depends on its shape and size (architecture) as well as on the amount of bone in it (bone mineral "density" and content), treatments intended to prevent or cure these conditions should strengthen the above cortex, and absorptiometric studies should begin to account for both bone architecture and bone tissue content. "Bone anabolic" agents (parathyroid hormone and some prostaglandins) can make modeling add bone to normal and osteopenic skeletons, but when the treatment stops remodeling begins removing that bone. "Antiremodeling agents" (including estrogen and many bisphosphonates) can make remodeling tend to keep existing bone, but when such treatments stop remodeling usually resumes removing bone. Combining anabolic agents with antiremodeling agents offers an exceptionally promising prospect of effective prevention and cure of the above osteopenias. Practical problems make this approach not yet ready for human use, but it soon could be.

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Johansen

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DN PubMed ID: 8845585
TI Bisphosphonates and the treatment of bone disease in the elderly.
AU Johansen A; Stone M; Rawlinson P
CS Academic Department of Geriatrics, University of Wales College of Medicine, Cardiff.
SO Drugs & aging, (1996 Feb) Vol. 8, No. 2, pp. 113-26. Ref: 133
Journal code: 9102074. ISSN: 1170-229X.
CY New Zealand
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 199610
ED Entered STN: 6 Nov 1996
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AB Recognition of the way in which naturally occurring pyrophosphate affects bone metabolism has stimulated the development of a whole series of related compounds, the bisphosphonates. Although the precise mechanism of action of these compounds remains incompletely understood, they have been proven profoundly effective in clinical practice and already constitute a major advance in the therapy of conditions characterised by excessive bone resorption. Serious adverse effects are rare; however, mineralisation problems are a concern, particularly with etidronate. The reduction in bone turnover during prolonged treatment for osteoporosis is also a concern but as yet of uncertain clinical importance. The wide variation in potency of different bisphosphonates in inhibiting the resorption, mineralisation and turnover of bone will increasingly determine which agents are used in various clinical situations. Bisphosphonates are the treatment of choice in Paget's disease and hypercalcaemia of malignancy. They also appear to have potential to alter the course of metastatic bony disease, although the magnitude and clinical importance of any such effect remains unclear. Bisphosphonates show promise in the prevention and treatment of osteoporosis and increase bone mass in postmenopausal and steroid-induced osteoporosis. The effects of these drugs in other situations are less clear and the effects on fracture rates are not yet fully characterised. Optimal regimens have yet to be established but the long half-lives of these agents makes intermittent treatment a rational and convenient approach.